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Lethal and Sublethal Effects of Imidacloprid on Nicotine-Tolerant *Myzus nicotianae* and *Myzus persicae*

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Abstract: Bioassays of nicotine and imidacloprid against clones of *Myzus persicae* (Sulzer) and *Myzus nicotianae* (Blackman) from around the world demonstrated that some had low levels of resistance to both compounds. This was expressed not only as a reduced mortality, but more markedly as differential inhibition of feeding by imidacloprid concentrations in the parts per billion range. Such concentrations also reduced aphid fecundity by inhibiting the production and viability of nymphs, and this effect was more marked for susceptible aphids than for those showing reduced direct lethal and antifeedant effects.

Key words: insecticide resistance, fumigation, bioassay, aphid fecundity, antifeedant

1 INTRODUCTION

Resistance to insecticides is now widespread in the peach-potato aphid,[1] *Myzus persicae* (Sulzer), and its tobacco-feeding form, *M. nicotianae* (Blackman). Although they have been considered to be distinct species,[2] they can interbreed and produce fertile eggs. Both rely on the same biochemical and genetic mechanisms for resistance to a wide range of carbamate, organophosphorus and pyrethroid insecticides. The most common is the overproduction of esterases (E4 or FE4) that detoxify insecticidal esters by sequestration and hydrolysis.[3] In addition, a new resistance mechanism has recently been discovered in both species based on insensitivity of acetylcholinesterase to pirimicarb and the novel aphicide triazamate.[4]

The need for a novel class of insecticide to circumvent these resistance mechanisms appears to be met by the new insecticide, imidacloprid, a nitroguanidine in the chloronicotinyl group related to the heterocyclic nitromethylenes, which has a mode of action similar to that of nicotine, i.e. acting as an agonist to acetylcholine at the nicotinic acetylcholine receptor.[5,6] This mode of action, together with its novel chemical structure render it unaffected by the established resistance mechanisms, so that it gives excellent control of conventionally resistant aphids.[7] It is also cited as being particularly effective in virus control because it disrupts feeding by homopterous insects.[7–10]

One possible source of resistance to this novel compound is target-site-based cross-resistance afforded to nicotine-tolerant insects. Nicotine tolerance has been reported in the tobacco hornworm *Manduca sexta* (Joh)[11,12] and the tobacco budworm *Heliothis virescens* F.[13] In this latter species, nicotine insensitivity correlates with elevated levels of cytochrome P-450.

As a tobacco-feeding species, *M. nicotianae* appears to avoid exposure to the nicotine-containing xylem.[14] However, some exposure to nicotine seems likely during adaptation over many years, with possible selection for aphids with a modification of the nicotinic acetylcholine receptor.

The present work examined the responses of *Myzus* spp. to nicotine fumigation and to imidacloprid treatment, to identify any possible correlations between the two. The requirements were for a simple screening assay with which to make rapid assessments of field (and reference) clones and for more complex assays which

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would allow the sublethal effects (e.g. decreased fertility and the production of non-viable offspring)[8,10] of imidacloprid to be made apparent.

2 METHODS AND MATERIALS

2.1 Aphids

Clones of both species were collected from a wide geographic area (Table 1). All these reference clones tested had been classified as *M. persicae* or *M. nicotianae* by Dr R. L. Blackman of the British Natural History Museum and screened *in vitro* for esterase content,[15] glutamate-oxaloacetate transaminase (GOT) allozymes,[16] and pirimicarb-insensitive AChE.[4]

All aphids and bioassays were maintained on chinese cabbage (*Brassica pekinensis* (Lour.) Rupr.)[17] at 22°C 16 : 8 h light : dark, except during nicotine fumigation when the temperature was 30 (±1)°C.

2.2 Bioassays

Nicotine fumigation and imidacloprid feeding-assay results represent the pooled data of three separate bioassays, whilst imidacloprid aphid-dip results represent the pooled data of at least two separate bioassays. Each bioassay consisted of three replicates of 10 adult apterous aphids for each clone and dose. Probit analysis was performed on the data using POLO (LeOra software).

2.2.1 *Nitocine: fumigation*

Ten aphids of each clone to be tested were placed on chinese cabbage leaf-discs, maintained on agar, in plastic tubs covered with gauze to prevent escape but allow fumigation.

Aqueous nicotine solution (8 μl; four concentrations and containing 100 ml litre^{-1} acetone) were spread on watch glasses placed inside 4-litre glass desiccators. The resulting nominal nicotine vapour concentrations (assuming complete evaporation) were calculated to be 0·008, 0·026, 0·064 and 0·26 mg litre^{-1} of air, i.e. not exceeding the saturation pressure of nicotine vapour (approx 0·45 mg litre^{-1}).

The caged aphids were immediately placed on a perforated wire shelf above the watch glass, and the desiccators sealed. They were placed in an incubator at 30 (±1)°C to facilitate vaporisation of the nicotine, and mortality was assessed after 24 h.

2.2.2 *Imidacloprid: aphid dips*

Aphids were exposed by the FAO-recommended aphid dip-test[18] to dilute solutions of technical imidacloprid which had been dissolved in acetone (the final acetone content for all imidacloprid concentrations was 10 ml litre^{-1}). After dipping, aphids were transferred to chinese cabbage leaves and scored after 43 h to give an EC_{50} for 'mortality' that combined death and irreversible symptoms.

2.2.3 *Imidacloprid: feeding assays*

The petioles of excised chinese cabbage leaves (approximately 25 cm^2 were placed in dilute aqueous

TABLE 1
Details of Laboratory-Reared Reference Clones

Clone	*Species*[a]	*Origin*	*Crop*	*E4/FE4*[b]	*AChE*[c]	*GOT*[d]
US1L	*M. persicae*	UK	Sugar beet	S	S	M.p.
794J	*M. persicae*	UK	Chrysanths	E4/R_3	S	M.p.
926B	*M. persicae*	Greece	Peach	E4/R_3	I	M.n.
927B	*M. nicotianae*	Greece	Peach	E4/R_3	S	M.p.
934E	*M. nicotianae*	USA	Tobacco	FE4/R_1	S	M.n.
935D	*M. nicotianae*	USA	Tobacco	S	S	M.n.
975A	*M. nicotianae*	Hungary	Potato	FE4/R_1	S	M.n.
1051A	*M. persicae*	Japan	Spinach	E4/R_2	I	M.p.
1052A	*M. nicotianae*	Japan	Sesame	S	S	M.p.
1053A	*M. nicotianae*	Japan	Potato	E4/R_2	S	M.p.
1054A	*M. persicae*	Japan	Cabbage	E4/R_3	I	M.p.
1074E	*M. persicae*	Germany	Cabbage	S	S	M.p.
1172A	*M. persicae*	Holland	Sweet Pepper	S	I	M.p.
1173C	*M. persicae*	Holland	Sweet Pepper	S	I	M.p.

[a] Formally identified by R. L. Blackman.[2]
[b] Aphids classified as susceptible (S), moderately (R_1), very (R_2) or extremely (R_3) resistant to established aphicides according to esterase (E4 or FE4) content.[15]
[c] Aphids classified as having pirimicarb-insensitive or -sensitive acetylcholinesterase.[4]
[d] Aphids had GOT allozymes[16] typical of, though not invariably found in, *Myzus persicae* (M.p.) or *Myzus nicotianae* (M.n.).

TABLE 2
Response of Aphids to Nicotine in Fumigation Bioassays

Clone	*Species*	LC_{50} [a] (*mg litre^{-1} air*)	*95% CL*[b]	*Slope*	*TF*[c]
US1L	*M. persicae*	0·023	0·016–0·030 **a**	1·98	1
794J	*M. persicae*	0·028	0·022–0·035 **a**	1·57	1
926B	*M. nicotianae*	0·12	0·091–0·168 **b**	1·67	5
927B	*M. nicotianae*	0·12	0·089–0·174 **b**	1·12	5
934E	*M. nicotianae*	0·12	0·087–0·165 **b**	1·36	5
975A	*M. nicotianae*	0·12	0·076–0·208 **b**	1·01	5
1051A	*M. persicae*	0·079	0·054–0·123 **b**	1·59	3
1053A	*M. nicotianae*	0·029	0·019–0·043 **a**	2·56	1
1054A	*M. persicae*	0·078	0·057–0·114 **b**	2·28	3

[a] Nominal lethal concentration (see text).
[b] 95% confidence limits; values followed by the same letter do not differ significantly ($P < 0.05$).
[c] Tolerance factor = LC_{50} for clone/LC_{50} for US1L.

solutions of imidacloprid 200 g litre^{-1} SL for 24 h after which time they were removed and placed in Blackman boxes resting in tap water.[17] Ten adult or 4th-instar aphids were immediately placed on each confined leaf and assessed after an additional 24 h.

Aphids were scored as probing, unsettled or dead; probing aphids were assumed to be feeding. The vast majority of aphids that were not probing later died, but data from a simple assessment of mortality gave poor probit lines. Dead and unsettled aphids were therefore added together to give a measure of the EC_{50} (effective concentration) needed to prevent feeding by *Myzus* spp.

2.2.4 Imidacloprid: nymph production
The petioles of eight excised chinese cabbage leaves were dipped in dilute aqueous solutions of imidacloprid for 24 h. They were then transferred to Blackman boxes and 10 aphids of each clone were placed on each leaf. After 60 h, the numbers of nymphs produced were counted, these being divided into viable and non-viable (with unformed limbs, antennae and cornicles). The percentage of non-viable nymphs being produced was determined and general decreases in nymph production were monitored.

3 RESULTS

3.1 Nicotine: fumigation

Although the nicotine vapour concentration in the desiccators was not measured, the bioassay conformed well to the probit model (with slopes between 1 and 2·5, Table 2) based on the concentration of nicotine solution

TABLE 3
Imidacloprid Aphid-Dip Bioassays with Reference Clones

Clone	*Species*	EC_{50} [a] (*mg AI litre^{-1}*)	*95% CL*[b]	*Slope*	*TF*[c]	*Nicotine TF*[d]
US1L	*M. persicae*	2·2	1·9–2·5 **a**	2·0	1·0	1
1051A	*M. persicae*	3·7	2·7–4·9 **b**	1·6	1·7	3
1074E	*M. persicae*	1·8	1·3–2·4 **a**	2·0	0·8	n.t.
927B	*M. nicotianae*	7·7	5·7–9·8 **c**	2·4	3·5	5
935D	*M. nicotianae*	6·7	4·7–9·1 **bc**	1·9	3·0	n.t.
975A	*M. nicotianae*	7·2	5·2–9·5 **c**	1·9	3·3	5
1052A	*M. nicotianae*	4·2	2·8–5·8 **bc**	1·8	2·2	n.t.
1053A	*M. nicotianae*	1·5	1·0–1·9 **a**	2·4	0·7	1

[a] Effective concentration to give 50% dead or with irreversible symptoms of poisoning.
[b] 95% confidence limits; values followed by the same letter do not differ significantly ($P < 0.05$).
[c] Tolerance factor = LC_{50} for clone/LC_{50} for US1L.
[d] Data taken from Table 2 for ease of comparison (n.t., not tested).

added, so confirming the utility of this bioassay for comparing strains.

Nicotine tolerance among *M. nicotianae* clones might be expected for a species adapted to feed on tobacco, but it is notable that some *M. persicae* clones originating from other hosts also exhibited this reduced response (1051A, 1054A, Table 2), i.e. there is resistance within, as well as tolerance between, the species. Nicotine tolerance is independent of any other resistance mechanism described in *Myzus* species, as is illustrated by comparing Tables 1 and 2, in which representatives from across the range of esterase content and AChE sensitivity show all degrees of tolerance. Clones 934E and 926B, for example, have very different FE4/E4 and AChE profiles, but are equally nicotine-tolerant.

3.2 Imidacloprid: aphid dips

For the reference clones, the EC_{50} values of imidacloprid for *M. persicae* and *M. nicotianae* cover a range of doses (1·8–7·7 mg litre^{-1}, Table 3). Many of these differences are significant, although the factors involved are typically only two- to three-fold based on the response of the most susceptible clone. It is notable that, as with nicotine, the responses of *M. persicae* clone 1051A and *M. nicotianae* clone 1053A to imidacloprid were atypical of their species. In fact, the LD_{50} values from nicotine fumigation assays broadly predicted the response of clones in the imidacloprid bioassays (final columns, Tables 3 and 4).

3.3 Imidacloprid: feeding assays

Imidacloprid's anti-feedant effect was expressed at very low concentrations indeed (US1L; $EC_{50} = 5{\cdot}82$ μg litre^{-1}, Table 4). There was, in comparison with other European *M. persicae*, an approximately 20-fold reduced response exhibited by two nicotine-tolerant strains of *M. nicotianae* (926B and 934E), and a single Japanese clone (*M. nicotianae;* 1053A) showed an ultra-susceptible response, so extending the range of tolerance to approximately 80-fold.

3.4 Imidacloprid: nymph production and viability

The total number of nymphs produced decreased with imidacloprid treatment (Table 5a), even at doses where no adults were killed and most were unaffected in terms of feeding behaviour (Tables 3 and 4).

The data (Table 5b) suggest that the effect of imidacloprid on nymph viability is also related to the tolerance to nicotine shown by a clone. 926B and 934E produced few, if any, non-viable nymphs (0–7%) in comparison to US1L and 794J (13–24%). It has been shown (G. Devine, unpublished results) that such tolerant aphids will also produce non-viable nymphs if the concentration of imidacloprid is increased, but the data presented show that imidacloprid can exert sublethal effects on susceptible aphids at the extremely low concentration of 0·2 μg litre^{-1}.

4 DISCUSSION

The severity of imidacloprid's lethal and sublethal effects on *Myzus* species is related to nicotine tolerance. The low level of tolerance to imidacloprid's direct lethal effect does not appear to be undermining its efficacy in practice.[19] However, nicotine appears to have only a small margin of 'overkill' in normal use that is partially overcome by the 4–5-fold tolerance, so that such aphids can survive fumigation with nicotine (unpublished observations).

At very low concentrations (6 μg litre^{-1}), imidacloprid can have a profound effect upon feeding behaviour in *M. persicae* and *M. nicotianae*, which would presumably afford a large degree of protection to plants under threat from some viruses. Nauen[10] also found imidacloprid to be very effective in preventing feeding; typically, a dose of 16 μg litre^{-1} depressed honeydew excretion to 50% of that in his control treatments. However, clones can exhibit a 50- to 80-fold range of sensitivity to imidacloprid based on this antifeeding criterion, even though there are only small differences in the direct lethal effect, and this would have implications for its protective effect against some virus diseases.

At lower concentrations still, the fecundity of aphids on an imidacloprid-treated leaf was markedly decreased (Tables 5a and 5b). For susceptible clones, treatment with 2 μg litre^{-1} imidacloprid resulted in approximately 30–50% of the nymph production compared with controls, and an additional 13–24% reduction in the viability of those nymphs. At this concentration, only 1·4 viable nymphs per adult US1L [(15·9/10) × (1 − 0·13) from Tables 5a and 5b] were produced in 60 h as opposed to 5·2 in the untreated controls.

Nicotine- and imidacloprid-tolerant clones are less susceptible to these effects on fecundity: 926B neither displayed decreased fertility nor produced non-viable nymphs at the same dose, and the proportion of non-viable nymphs produced by 934E is markedly less than that for either of the nicotine-susceptible clones (US1L and 794J).

Biotypes of *Myzus* species clearly have the potential to develop multiple resistance mechanisms. Although the biochemical roles of E4 and FE4 esterases[3] and of insensitive AChE[4] are well established, the apparent cross-resistance between nicotine and imidacloprid provides, as yet, only circumstantial evidence that this new mechanism is based on target-site insensitivity.

It is impossible, by laboratory bioassay alone, to quantify the severity of resistance in the field. Surveys of field populations of aphids using 'discriminating doses' of imidacloprid, based on the aphid dip-test results presented here, did not give any evidence that such toler-

TABLE 4
EC_{50} Values for Feeding Prevention by Imidacloprid

Clone	*Species*	*EC_{50}* [a] *(μg AI litre^{-1})*	*95% CL*[b]	*Slope*	*TF*[c]	*Nicotine TF*[d]
US1L	*M. persicae*	5·8	2·4–11·7 **a**	0·795	1	1
794J	*M. persicae*	2·9	0·92–7·38 **ab**	0·775	0·5	1
1053A	*M. nicotianae*	0·68	0·36–1·16 **b**	0·635	0·1	1
926B	*M. nicotianae*	53	34·6–79·6 **c**	0·813	9	5
934E	*M. nicotianae*	35	23·2–53·6 **c**	0·711	6	5
1173A	*M. persicae*	3·10	1·08–6·24 **ab**	0·98	0·5	n.t.
1172C	*M. persicae*	6·7	2·94–13·2 **a**	0·653	1	n.t.

[a] Effective concentration to give 50% dead or not feeding.
[b] 95% confidence limits; values followed by the same letter do not differ significantly ($P < 0·05$).
[c] Tolerance factor = LC_{50} for clone/LC_{50} US1L.
[d] Data taken from Table 2 for ease of comparison (n.t., not tested).

ance is compromising the performance of imidacloprid against field populations.[19] Although recommended field dose rates are in the lethal range, the very marked differential in fecundity, which favours the tolerant variants, has major implications for their build-up in aphid populations, especially as residues decline.

Monitoring the incidence of imidacloprid resistance in the field is therefore critical to resistance management in these *Myzus* species. Of the clones studied here, nicotine and imidacloprid tolerance were predominantly, though not exclusively, associated with *Myzus nicotianae*. However, the demonstration that there has been

TABLE 5a
Number (Mean and SE) of Nymphs Produced by Groups of Ten Aphids during 60 h Exposure to Imidacloprid-Treated Leaves

Species	*Clone*	*Dose (μg litre^{-1})*[a] *0*	*0·2*	*2*
M. persicae	US1L	51·9 (±4·4) **a**	14·8 (±3·2) **b**	15·9 (±4·2) **b**
M. persicae	794J	29·4 (±2·8) **a**	22·4 (±1·8) **a**	15·1 (±1·4) **b**
M. nicotianae	926B	31·9 (±4·4) **a**	27·9 (±1·3) **a**	27·0 (±2·9) **a**
M. nicotianae	934E	24·8 (±1·8) **a**	23·1 (±4·0) **ab**	12·7 (±3·0) **b**

[a] Different letters denote significant ($P < 0·05$) differences between doses for a given clone.

TABLE 5b
Fraction (Mean and SE) of Nymphs that were Non-viable

Species[a]	*Clone*	*Dose (mg litre^{-1})*[b] *0*	*0·2*	*2*
M. persicae	US1L	0·00 **a**	0·20 (±0·03) **b**	0·13 (±0·03) **b**
M. persicae	794J	0·00 **a**	0·19 (±0·02) **b**	0·24 (±0.03) **b**
M. nicotianae	926B	0·00	0·00	0·00
M. nicotianae	934E	0·00 **a**	0·08 (±0·02) **b**	0·07 (±0·03) **b**

[a] *M. nicotianae* clones produced fewer non-viable nymphs in response to treatment than *M. persicae* clones ($P < 0·05$).
[b] Different letters denote significant ($P < 0·05$) differences between doses for a given clone.

introgression of esterase-based resistance between *M. persicae* and *M. nicotianae*[20] warns that this problem is unlikely to remain associated predominantly with the tobacco-feeding variant.

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